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# Review on Drug Induced Nephrotoxicity and Therapeutic Strategies

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**ABSTRACT**

Drug-induced nephrotoxicity represents a significant clinical challenge, contributing to acute kidney injury (AKI), and chronic kidney disease (CKD). The kidney's high exposure to circulating drug makes them vulnerable to toxic injury through mechanism such as direct tubular toxicity, hemodynamic alterations, immune-mediated reactions, crystal deposition, rhabdomyolysis, thrombotic microangiopathy, and oxidative stress. Heavy metals and certain herbal agents further exacerbate renal damage. Early detection using biomarkers like NGAL(Neutrophil Gelatinase Associated Lipocalin) and KIM-1(Kidney Injury Molecule-1) offers improved sensitivity compared to traditional creatinine monitoring. Preventive strategies include hydration therapy, dose adjustment, and renal function monitoring, while therapeutic approaches emphasize antioxidants, hemodynamic modulators, and natural bioactive compounds. Emerging interventions such as mesenchymal stem cell therapy and remote ischemic preconditioning show promise in mitigating renal injury. Collectively, these strategies highlight the importance of integrating pharmacological and non-pharmacological measures to reduce nephrotoxicity and improve patient outcomes.

**Keywords**: Nephrotoxicity; Drug-induced kidney injury; Biomarkers; Nephroprotection.

## INTRODUCTION

Nephrotoxicity is defined as kidney damage or impaired renal function that arises from exposure to harmful agents such as drugs, chemicals, or environmental toxins. Clinically, it is important because, if not identified early, it can progress to acute kidney injury (AKI), chronic kidney disease (CKD), or even irreversible renal failure [1]. Nephrotoxicity describes the harmful impact of certain substances on the kidneys, leading to damage in renal structure or disruption of normal kidney function[2].

Nephrotoxicity develops when harmful agents such as drugs, chemicals, or naturally occurring toxins disrupt normal kidney physiology. This disruption may occur through direct injury to renal tubules, alteration in renal blood flow, or activation of immune-mediated mechanism that damage renal tissue [3]. The Clinical Context disposes many therapeutic agents (e.g., aminoglycosides, NSAIDs, Antineoplastic agents, Heavy metals, radiocontrast agents etc..) are known to cause nephrotoxicity, especially in patients with pre-existing renal disease or other risk factors [4].

Recent evidence from 2024–2025 indicates that drug-induced nephrotoxicity continues to be a major cause of acute kidney injury , contributing to approximately 8–60% of cases worldwide. Large cohort studies further demonstrate that medications are implicated in 14–26% of AKI episodes among adults and in nearly 16% of pediatric cases, underscoring the increasing clinical burden associated with drug-related renal injury [5]. Drug development is frequently hindered by renal safety concerns, as nephrotoxicity accounts for approximately 8–9% of failures in both preclinical and clinical trials. This makes drug-induced kidney injury a significant obstacle in pharmaceutical innovation and a critical factor in the evaluation of candidate compounds [6].

Drug-induced nephrotoxicity is a leading cause of acute kidney injury (AKI) among hospitalized patients. When recognized promptly, discontinuation or removal of the offending agent often allows renal function to recover. However, prolonged or repeated exposure can result in irreversible damage, ultimately progressing to chronic kidney disease (CKD) [7]. Oxidative stress contributes significantly to kidney damage through several mechanisms. These include lipid peroxidation of tubular cell membranes, oxidative modification of key renal enzymes, and direct DNA injury. Such molecular alterations can trigger pathways leading to apoptosis or necrosis, ultimately compromising renal structure and function[8].

Antioxidants serve an essential protective function in the kidney by neutralizing reactive oxygen species and reducing oxidative stress. Through this mechanism, they help preserve cellular integrity, limit lipid peroxidation, and prevent oxidative damage to

proteins and DNA, thereby mitigating the progression of nephrotoxicity [9], scavenging reactive oxygen species, Inhibition of lipid peroxidation, protection of proteins and enzymes, and preservation of DNA integrity[10]. Risk elements comprise older age, lack of hydration, diabetes, high blood pressure, and multiple medications [11].

Regular assessment of kidney function is essential when prescribing drugs with nephrotoxic potential. Standard monitoring includes measurement of serum creatinine, blood urea nitrogen (BUN), and evaluation of urine output. These tests provide early indicators of renal impairment, allowing timely intervention to prevent progression of drug-induced nephrotoxicity [12]. Timely identification and prevention of nephrotoxicity are crucial, as drug-induced kidney injury is often reversible when recognized promptly. Early withdrawal of the offending agent can restore renal function, whereas delayed diagnosis or prolonged exposure increases the risk of progression to chronic kidney disease (CKD) and is associated with higher mortality [13].

Preventing drug-induced nephrotoxicity requires proactive measures. Risk assessment identifies vulnerable patients, while dose adjustment minimizes exposure. Adequate hydration supports renal perfusion, and routine monitoring of kidney function through laboratory tests enables early detection. Together, these strategies significantly lower the incidence and burden of drug-related renal injury in clinical practice [14].

Emerging biomarkers improve early detection of nephrotoxicity. NGAL, measurable in plasma and urine, identifies acute kidney injury within hours. KIM-1 serves as a specific marker of tubular damage, offering high diagnostic accuracy. In contrast, serum creatinine rises later and is less sensitive, making newer markers superior for timely recognition [15].

**Mechanisms of Drug-Induced Nephrotoxicity**

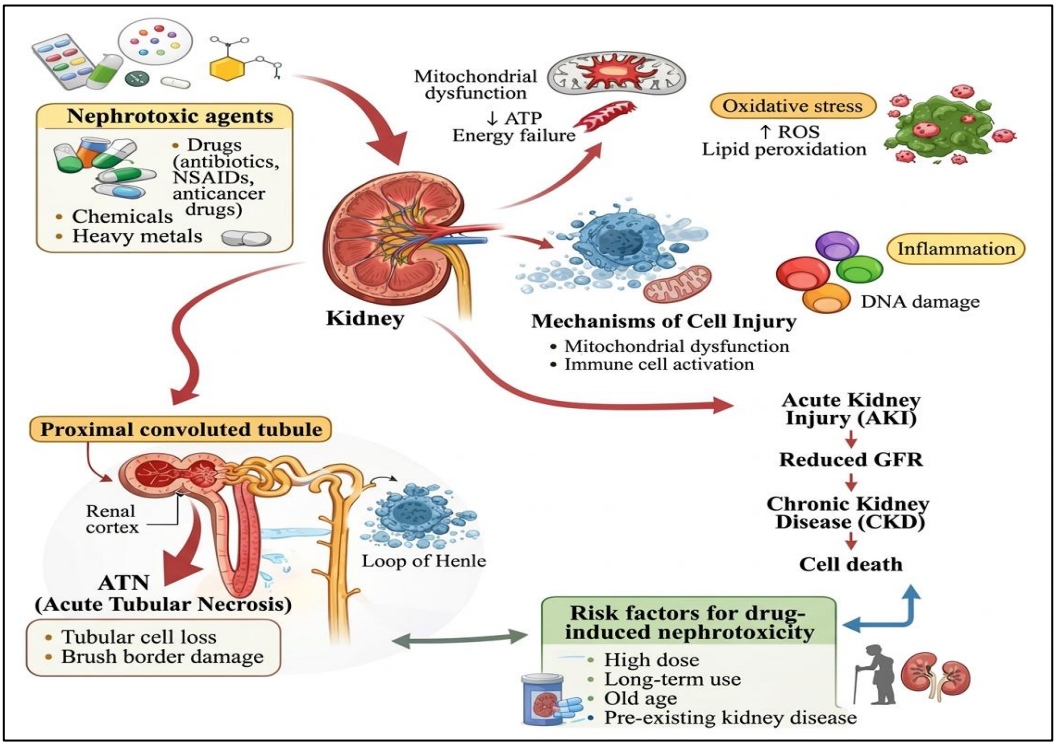


**Fig 1: Mechanism of Drug-induced Nephrotoxicity**

Nephrotoxicity arises when medications or their metabolites inflict structural or functional injury on the kidneys. Because the kidneys filter large volume of blood and concentrate substances within the renal tubules, they are particularly susceptible to toxic insults, making them a common target of drug-related damage [1].

**Direct Tubular Toxicity**

Certain medications, including aminoglycosides, cisplatin, and amphotericin B, tend to accumulate within renal tubular epithelial cells. Their presence induces oxidative stress, disrupts mitochondrial function, and activates cell death pathways such as apoptosis. These processes collectively contribute to acute tubular necrosis (ATN), a major manifestation of drug-induced nephrotoxicity [17].

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Approximately one-third of pediatric patients exposed to nephrotoxic drugs develop proximal tubular dysfunction. This impairment can progress to Renal Fanconi Syndrome (RFS), a condition characterized by defective reabsorption in the proximal tubules, leading to significant disturbances in electrolyte balance and overall renal function [18].

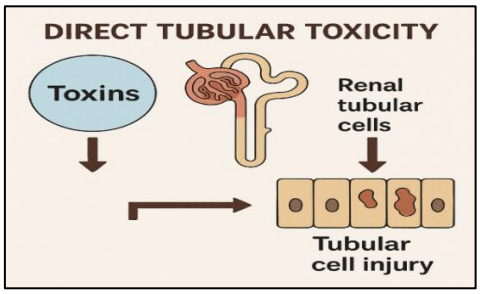


**Fig 2. Direct Tubular Toxicity**

**Hemodynamic Alteration**

Alterations in renal hemodynamics represent a major pathway of drug-induced kidney injury. These changes primarily affect renal blood flow and glomerular filtration. Agents such as nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and calcineurin inhibitors disrupt the balance between vasodilatory and vasoconstrictive mediators, thereby impairing renal perfusion [19]. Nonsteroidal anti-inflammatory drugs (NSAIDs) reduce prostaglandin synthesis, limiting afferent arteriolar dilation and thereby decreasing glomerular blood flow. In contrast, angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) inhibit angiotensin II–mediated efferent arteriolar constriction, which lowers intraglomerular pressure and can compromise filtration efficiency [20].

Calcineurin inhibitors enhance vasoconstrictor activity, further impairing renal circulation. These hemodynamic disturbances reduce glomerular filtration rate (GFR) and predispose patients to acute kidney injury. In individuals with heightened susceptibility, persistent exposure may drive progression toward chronic kidney disease, underscoring the clinical importance of careful monitoring and dose regulation [21]. Drugs that alter vascular tone or interfere with autoregulatory mechanisms of renal blood flow can precipitate nephrotoxicity. By disrupting the balance of perfusion, these agents increase the risk of kidney injury. This underscores the importance of vigilant monitoring, particularly in individuals with heightened susceptibility to renal dysfunction [22].

**Immune-Mediated Injury (Acute Interstitial Nephritis)**

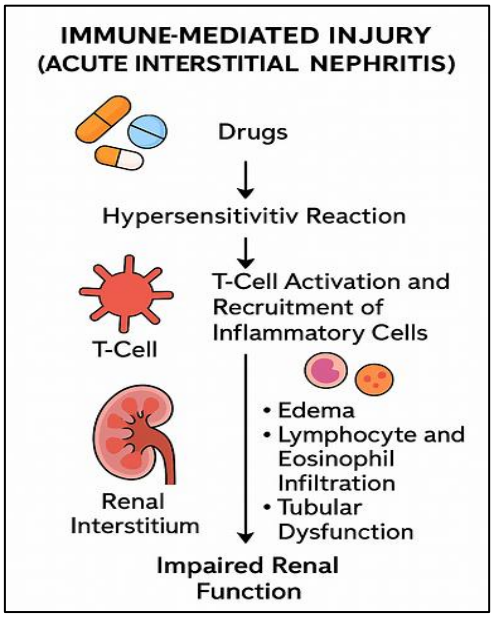


**Fig 3. Immune Mediated Injury**

Immune-mediated injury, most notably acute interstitial nephritis (AIN), is a common mechanism of drug-induced nephrotoxicity. It develops when medications such as antibiotics, NSAIDs, or proton pump inhibitors provoke a hypersensitivity reaction that activates T-cells and recruits inflammatory cells into the renal interstitial [23]. This immune cascade causes oedema, infiltration of lymphocytes and eosinophils, and tubular dysfunction, ultimately impairing renal function [24].The underlying pathogenesis involves drug-related modification of tubular antigens or hapten formation, which triggers an immune attack against kidney tissue. Prompt identification and withdrawal of the causative drug are essential to prevent chronic damage, and corticosteroids may be considered to hasten recovery in selected cases [25].

**Crystal-Induced Obstruction**

Crystal-induced obstruction is a recognized mechanism of drug-related nephrotoxicity, occurring when poorly soluble drug metabolites precipitate within renal tubules and block urine flow [26]. Agents such as acyclovir, indinavir, methotrexate, and sulphonamides can form crystals under conditions of high concentration or low urinary pH, leading to tubular obstruction, increased intratubular pressure, and subsequent decline in glomerular filtration [27]. The resulting mechanical blockage triggers local inflammation and oxidative stress, further aggravating renal injury. Recent studies emphasize that adequate hydration, urine alkalinization, and dose adjustment are key strategies to prevent crystal-induced nephropathy [28].

**Rhabdomyolysis-Associated Nephrotoxicity**

Certain medications and substances can damage skeletal muscle cells (myocytes) either through direct toxic effects or by increasing their susceptibility to stress, such as during exercise. When these muscle cells are injured, they undergo lysis, releasing intracellular components like myoglobin and creatine kinase into circulation [29]. Myoglobin, in particular, can be harmful to the kidneys due to its intrinsic toxicity and its tendency to obstruct renal tubules, leading to kidney injury. Agents such as statins, excessive alcohol intake, heroin, ketamine, and cocaine have all been associated with rhabdomyolysis, a condition characterized by muscle breakdown and subsequent renal complications [30]. Statins and certain antivirals may cause muscle breakdown. Myoglobin released into circulation accumulates in renal tubules, causing oxidative damage and obstruction [31].

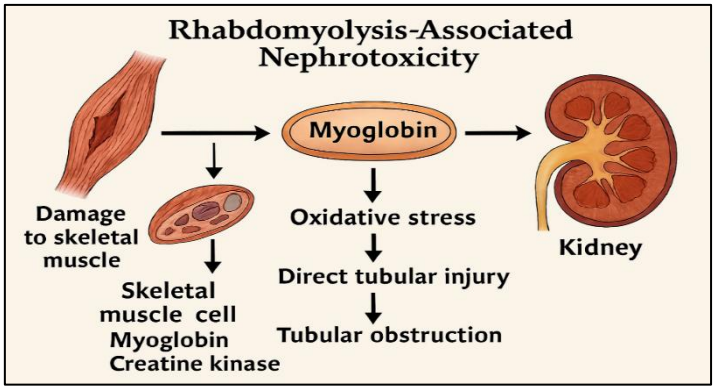


**Fig 4: Rhabdomyolysis-Associated Nephrotoxicity**

**Thrombotic Microangiopathy**

Thrombotic microangiopathy (TMA) represents a critical pathway of drug-induced nephrotoxicity, marked by endothelial cell injury, platelet aggregation, and microvascular thrombosis within the kidney[32]. Medications such as calcineurin inhibitors (cyclosporine, tacrolimus), chemotherapeutic agents (gemcitabine, mitomycin C), and anti-VEGF drugs can provoke endothelial dysfunction, leading to narrowing of renal microvessels, hemolysis, and impaired perfusion[33]. The formation of microthrombi within glomerular capillaries results in acute kidney injury, proteinuria, and progressive decline in renal function[34]. Mechanistically, TMA involves complement activation, oxidative stress, and direct endothelial toxicity. Early detection and discontinuation of the causative drug are essential to prevent irreversible renal damage, as highlighted in recent nephrology literature [35].

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**Oxidative Stress**

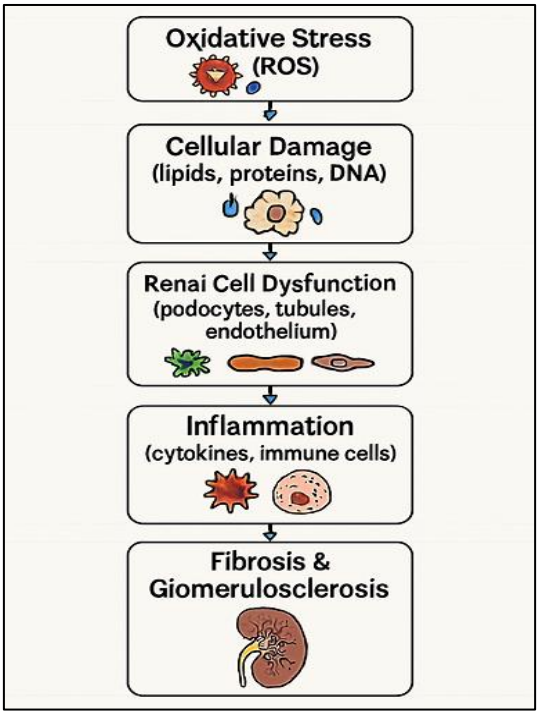


**Fig 5**: Oxidative Stress

Oxidative stress plays a central role in the pathogenesis of drug-related kidney injury, driving progressive renal damage [36]. Several nephrotoxic agents, including aminoglycosides, cisplatin, and cyclosporine, promote excessive generation of reactive oxygen species (ROS) within renal tubular cells. This imbalance between oxidants and antioxidants contributes to cellular dysfunction and tissue injury [36]. Excessive reactive oxygen species (ROS) overwhelm the kidney's antioxidant defence systems, initiating lipid peroxidation, protein denaturation, and DNA damage. This oxidative imbalance disrupts mitochondrial integrity, diminishes ATP generation, and activates cell death pathways. As a result, renal tubular cells undergo apoptosis or necrosis, driving progressive drug-induced kidney injury [37]. Oxidative stress not only drives cellular injury but also activates inflammatory signalling and promotes fibrotic changes, further compromising renal structure and function. Recent evidence suggests that therapeutic strategies targeting oxidative stress—such as antioxidants or mitochondrial protectors—can attenuate drug-induced renal damage. Thus, oxidative stress represents a central pathogenic pathway in nephrotoxicity, underscoring the importance of early detection and protective interventions [38].

**Table 1: Mechanisms of Drug-Induced Nephrotoxicity.**

| Mechanism | Example Drugs | Pathology |
|---|---|---|
| Direct tubular toxicity | Aminoglycosides, cisplatin | Acute tubular necrosis |
| Hemodynamic changes | NSAIDs, ACE inhibitors | Ischemic injury, ↓ GFR |
| Immune-mediated | Penicillins, PPIs | Acute interstitial nephritis |
| Crystal deposition | Acyclovir, methotrexate | Tubular obstruction |
| Rhabdomyolysis | Statins, antivirals | Myoglobin-induced tubular injury |
| Microangiopathy | Gemcitabine, quinine | Endothelial damage, thrombosis |

**Heavy Metals Renal Toxicity:**

Heavy metals Renal toxicity Mercury Damage to the renal proximal tubule and heme-biosynthetic pathways Lead Chronic interstitial nephritis Arsenic Renal tubular necrosis; tubular degeneration; lymphomonocytic infiltration [39].

**Table 2: Heavy Metals and Renal Toxicity**

| Heavy Metal | Primary Renal Effect | Pathological Features |
|---|---|---|
| Mercury | Damage to renal proximal tubules | Disruption of tubular reabsorption; interference with heme biosynthetic pathways[39] |
| Lead | Chronic interstitial nephritis | Progressive interstitial fibrosis; tubular atrophy; long-term decline in renal function[40] |
| Arsenic | Renal tubular necrosis | Tubular degeneration; lymphomonocytic infiltration; impaired urine concentration [41] |

**Common Herbal Agents Implicated in Nephrotoxicity**

The growing popularity of herbal remedies has paralleled a rise in kidney-related disorders. Although many plant-based products can be safe when used appropriately under professional guidance, the increasing trend of self-medication, complex polyherbal mixtures, and widely marketed dietary supplements without medical supervision has heightened the risk of harmful renal effects. This concern is especially pronounced in low-resource regions, where access to conventional healthcare is limited and regulatory control over herbal product manufacturing is often weak [42].

Several herbal agents have been associated with diverse nephrotoxic pathways, including direct tubular injury, immune-mediated glomerular inflammation, oxidative stress, ischemic damage, and crystal deposition within the kidneys. Plants such as *Aristolochia* species, *Tripterygium wilfordii*, and *Glycyrrhiza glabra* have been repeatedly highlighted in case reports and clinical investigations. Among these, aristolochic acid stands out as a highly potent renal toxin and carcinogen, capable of inducing both acute and chronic kidney disease [43].

Moreover, many traditional and Ayurvedic preparations have been found to contain high levels of heavy metals like lead, mercury, and arsenic—either as intentional components based on historical texts or due to environmental contamination. These metals accumulate in renal tissue, leading to interstitial fibrosis and permanent damage [42].

**Table 3: Common Herbal Agents Implicated in Nephrotoxicity**

| Herbal Agent | Toxic Component | Associated Renal Effect |
|---|---|---|
| Dutchman's Pipe . | Aristolochic acid | Interstitial fibrosis, CKD |
| Thunder God Vine | Triptolide | AKI, tubular necrosis |
| Ghritkumari (high doses | Anthraquinones | Tubulointerstitial nephritis |
| Yashtimadhu | Glycyrrhizin | Hypokalemia, rhabdomyolysis, AKI |
| Teucrium chamaedrys | Neoclerodane diterpenes | Hepatorenal syndrome |

**Clinical Presentation**

The extent of kidney damage and the specific plant involved. In the initial stages, symptoms are often vague, such as tiredness, reduced urine production, and a general sense of malaise—signs that overlap with many renal conditions [44]. As the injury progresses, patients may develop swelling (edema), high blood pressure, and disturbances in electrolytes like potassium and sodium. In more severe cases, renal function can decline abruptly, resulting in acute kidney injury (AKI)[45]. This stage is marked by a sudden rise in serum creatinine, reduced urine output (oliguria), and fluid retention, which can become life-threatening if not recognized and treated quickly. Long-term or repeated exposure to harmful herbs may eventually cause chronic kidney disease, and if left unmanaged, can progress to end-stage renal failure [46].

**Diagnosis**

Rodent studies investigating drug-induced kidney injury demonstrate pathological and biochemical changes that closely resemble those seen in humans [47]. Both experimental animals and exposed individuals show increased levels of serum creatinine, blood urea nitrogen, and uric acid, indicating impaired renal function. Histological examinations consistently reveal structural damage, including tubular injury and fibrotic changes, highlighting in drug-related nephrotoxicity [48].

Several biochemical markers were evaluated, including KIM-1, NGAL, urea, blood urea nitrogen (BUN), glucose, creatinine, albumin, alanine aminotransferase (ALT), alkaline phosphatase (ALP), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and total protein (TP) [49,50]. Demographic information such as age and gender was also documented. Serum concentrations of KIM-1 and NGAL were quantified using enzyme-linked immunosorbent assay (ELISA) kits obtained from

BTLAB (Shanghai Korain Biotech Co., Ltd). The absorbance was measured at 450 nm with a microtiter plate [51]. Levels of were analysed through standard colorimetric techniques using an automated analyser (Roche Modular Autoanalyzer; Roche, Tokyo, Japan) Pharmacological Strategies [52].

**Table 4: Biomarker of clinical relevance**

| Biomarker / Parameter | Primary Role / Clinical Relevance |
|---|---|
| KIM-1 (Kidney Injury Molecule-1) | Sensitive marker of proximal tubular injury; useful in detecting drug-induced nephrotoxicity [53]. |
| NGAL (Neutrophil Gelatinase-Associated Lipocalin) | Early biomarker of acute kidney injury (AKI); rises within hours of tubular damage[54]. |
| Urea | End product of protein metabolism; elevated levels indicate impaired renal clearance[55]. |
| Blood Urea Nitrogen (BUN) | Reflects nitrogenous waste in blood; used to assess kidney function and hydration status[56]. |
| Glucose | Monitored for metabolic status; abnormal levels may affect renal function and overall homeostasis[57]. |
| Creatinine | Standard marker of glomerular filtration rate (GFR); rises later in AKI compared to NGAL/KIM-1[58]. |
| Albumin | Major plasma protein; low levels suggest proteinuria or impaired liver/kidney function[59]. |
| Alanine Aminotransferase (ALT) | Liver enzyme; elevated levels indicate hepatocellular injury, sometimes linked with systemic toxicity[60]. |
| Alkaline Phosphatase (ALP) | Enzyme linked to liver and bone function; abnormal values may indicate cholestasis or bone disease[61] |
| Aspartate Aminotransferase (AST) | Enzyme reflecting liver and muscle injury; elevated in systemic toxicity[62] |
| Gamma-Glutamyl Transferase (GGT) | Enzyme associated with liver function and oxidative stress; useful in hepatobiliary assessment [63] |
| Total Protein (TP) | Reflects overall protein status; changes may indicate nutritional status, liver disease, or renal loss[64] |

**Preventive & Therapeutic Strategies**

**Antioxidant**

N-acetylcysteine (NAC), vitamin C, vitamin E, flavonoids, polyphenols, and iron chelators represent key antioxidant strategies explored for nephroprotection. NAC acts as a glutathione precursor that strengthens intracellular defence, reduces contrast-induced acute kidney injury, and improves renal blood flow, though its clinical impact is limited by poor bioavailability [65]. Vitamin C, a potent free radical scavenger, not only mitigates cisplatin-induced oxidative damage but also regenerates vitamin E, which itself protects cell membranes from lipid peroxidation and shows synergistic effects when combined with vitamin C. Flavonoids and polyphenols such as quercetin and resveratrol suppress reactive oxygen species, modulate inflammation, and enhance endothelial function, with strong evidence in animal models though human data remain scarce[66,67]. Iron chelators like deferoxamine prevent iron-driven radical formation and reduce oxidative stress in drug-induced kidney injury, but their use is constrained by side effects and specific indications. Collectively, these antioxidants highlight promising avenues for reducing nephrotoxicity, though their effectiveness in clinical practice requires further validation [68].

**Table 5: Antioxidant Mechanism**

| Antioxidant | Mechanism | Evidence Strength | Limitations |
|---|---|---|---|
| NAC | Boosts glutathione, stabilizes NO | Moderate (RCTs, meta-analyses) | Poor bioavailability |
| Vitamin C | Free radical scavenger | Moderate (animal + small trials) | Dose-dependent efficacy |
| Vitamin E | Membrane protection | Moderate (experimental) | Works best in combination |
| Flavonoids | Anti-inflammatory, ROS reduction | Strong (animal studies) | Limited clinical trials |
| Iron Chelators | Prevent ROS via iron binding | Experimental | Side effects, narrow use |

**Hemodynamic Modulating Drugs**

Hemodynamic modulation is a key approach in nephroprotection, especially in chronic kidney disease. Agents such as ACE inhibitors and ARBs lower intraglomerular pressure, reduce proteinuria, and counteract hyperfiltration, thereby slowing disease progression [69]. Renin–angiotensin system blockade has long been established as standard therapy, while newer drugs like SGLT2 inhibitors provide both glycemic control and renal benefits by restoring tubuloglomerular feedback. Combining SGLT2 inhibitors with ACEIs or ARBs offers additive protection in diabetic kidney disease. In addition, endothelin receptor antagonists and vasodilatory agents are being explored for their ability to improve renal perfusion and limit ischemic injury [70]. Overall, these strategies emphasize the importance of optimizing renal blood flow and glomerular dynamics to preserve kidney function and delay progression to end-stage disease [71].

**Active Ingredients of Natural Medicines**

Active ingredients from natural medicines demonstrate significant nephroprotective potential through diverse mechanisms. Flavonoids such as quercetin and rutin, along with polyphenols like resveratrol and curcumin, act as strong antioxidants that neutralize reactive oxygen species and reduce lipid peroxidation in kidney tissues [72]. Saponins and terpenoids derived from plants including *Panax ginseng* and *Glycyrrhiza glabra* provide anti-inflammatory and cytoprotective benefits [73]. Alkaloids such as berberine enhance renal hemodynamics and regulate pathways linked to fibrosis. These compounds not only protect against drug-induced nephrotoxicity, including cisplatin and aminoglycoside damage, but also support chronic kidney disease management by lowering oxidative stress and preserving glomerular function [74]. Both experimental and clinical studies suggest that phytochemicals can serve as adjuncts to conventional therapies [75].

**Hydration Therapy**

Hydration therapy is one of the most effective and widely recommended strategies for nephroprotection, particularly in preventing contrast-induced acute kidney injury (CI-AKI) [76]. Adequate intravenous or oral fluid administration helps maintain renal perfusion, dilute nephrotoxic agents, and promote urine flow, thereby reducing tubular damage [77]. Clinical trials have consistently shown that isotonic saline hydration before and after contrast exposure significantly lowers the risk of CI-AKI compared to no hydration or hypotonic solutions [76]. In addition, hydration combined with adjunctive measures such as sodium bicarbonate infusion may further enhance renal protection. Overall, maintaining optimal hydration remains a cornerstone in safeguarding kidney function against iatrogenic injury [78].

**Remote Ischemic Preconditioning**

Remote ischemic preconditioning (RIPC) is increasingly recognized as a non-pharmacological intervention for nephroprotection, particularly against ischemia/reperfusion injury. The technique involves brief cycles of ischemia and reperfusion in a distant organ, such as the limb, which triggers systemic protective responses in the kidney [79,80]. Mechanistically, RIPC reduces oxidative stress, suppresses pro-inflammatory signalling pathways including TNF-α/NF-κB and TGF-β, and enhances nitric oxide bioavailability, thereby improving endothelial function and renal perfusion [81]. It also activates intracellular survival pathways such as PI3K/Akt and ERK1/2, which preserve mitochondrial integrity and limit apoptosis [82]. Recent experimental work in 2024 demonstrated that RIPC prevented renal ischemia/reperfusion injury in rats by modulating oxidative stress and inflammatory signalling, while a 2025 systematic review confirmed its potential to reduce renal injury in patients undergoing partial nephrectomy [83]. Collectively, these findings suggest that RIPC offers a promising adjunctive strategy for kidney protection, though further large-scale clinical validation is required [84].

**Haemodialysis**

Haemodialysis is not only a renal replacement therapy but also serves as a nephroprotective intervention by mitigating the harmful effects of toxin accumulation and fluid overload [85]. The mechanism of protection involves clearance of uremic solutes, regulation of acid–base balance, and stabilization of electrolytes, which collectively reduce oxidative stress and systemic inflammation [86]. By improving hemodynamic stability and reducing circulating pro-inflammatory mediators, haemodialysis helps preserve residual renal function and prevents further ischemic damage [87]. The cardiovascular complications associated with CKD, thereby indirectly supporting kidney protection through improved systemic circulation. Moreover, innovations such as biocompatible dialysis membranes and hemodiafiltration techniques have been shown to enhance toxin removal and reduce inflammatory responses, offering additional nephroprotective benefits [88]. Overall, haemodialysis remains a cornerstone in advanced kidney disease management, with evolving strategies aimed at maximizing renal protection and patient outcomes [89].

**Mesenchymal Stem Cell Therapy**

Mesenchymal stem cell (MSC) therapy has emerged as a promising approach in nephroprotection due to its ability to repair renal tissue and modulate harmful pathways [90]. MSCs exert protective effects by secreting paracrine factors that reduce oxidative stress, suppress pro-inflammatory cytokines, and enhance anti-inflammatory signalling, thereby limiting tubular and glomerular injury [91]. They also promote angiogenesis, improve mitochondrial function, and stimulate endogenous repair mechanisms, which collectively preserve renal structure and function. Recent studies have reported that MSCs attenuate ischemia/reperfusion injury and drug-induced nephrotoxicity, highlighting their potential as adjunctive therapy in both acute and chronic kidney disease [92]. While clinical translation is still evolving, MSC therapy represents a novel regenerative strategy for kidney protection [93].

**Conclusion**

Drug-induced nephrotoxicity remains a major contributor to renal morbidity and mortality worldwide. Its multifactorial mechanisms underscore the need for vigilant monitoring, risk assessment, and timely intervention. Advances in biomarker discovery have enhanced early detection, while therapeutic innovations ranging from antioxidants to stem cell therapy offer new avenues for nephroprotection. Preventive measures such as hydration therapy and hemodynamic modulation remain cornerstones of clinical practice. However, variability in patient susceptibility and limited clinical validation of novel therapies highlight the need for further research. Ultimately, a multidisciplinary approach combining pharmacological, non-pharmacological, and regenerative strategies is essential to minimize drug-related kidney injury and safeguard renal health in both acute and chronic disease.

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